IN THE CLAIMS

Please amend the claims as follows:

Please cancel claims 5-25, ~~37~~-40, ~~49~~-54, ~~57~~, ~~64~~-70 without prejudice.

Please add the following new claims 71-181:

71. The method of claim 1, wherein the RTA is a protease inhibitor.

72. The method of claim 1, wherein the RTA is a NRTI.

73. The method of claim 1, wherein the culture conditions comprise culturing the cell in the presence of a receptor ligand selected from the group consisting of a PPARγ ligand, a RXR ligand, a retinoic acid receptor ligand, insulin, an insulin-like growth factor, a glucocorticoid receptor ligand, and a cAMP-elevating agent.

74. The method of claim 73, wherein the receptor ligand is a PPARγ ligand.

75. The method of claim 74, wherein the PPARγ ligand is an agonist of PPARγ.

76. The method of claim 75, wherein the PPARγ agonist is a thiazolidinedione.

77. The method of claim 73 wherein the receptor ligand is a RXR ligand.

78. The method of claim 77, wherein the RXR ligand is an agonist of RXR.

79. The method of claim 78, wherein the RXR agonist is LGD1069, LG100268, 9-cis retinoic acid, or all-trans retinoic acid.

80. The method of claim 73, wherein the receptor ligand is a retinoic acid receptor ligand.

81. The method of claim 80, wherein the retinoic acid ligand is CH55, 9-cis retinoic acid, or all-trans retinoic acid.

82. The method of claim 73, wherein the receptor ligand is insulin.

83. The method of claim 73, wherein the receptor ligand is an insulin-like growth factor.

84. The method of claim 71, wherein the protease inhibitor is an aspartyl protease inhibitor.

85. The method of claim 84, wherein the aspartyl protease inhibitor is a viral aspartyl protease inhibitor.

86. The method of claim 85, wherein the viral aspartyl protease inhibitor is an HIV protease inhibitor.

87. The method of claim 72, wherein the NRTI is an HIV NRTI.

88. The method of claim 2, wherein the mesenchymal stem cell has the characteristics of a C3H10T1/2 cell.

89. The method of claim 88, wherein the mesenchymal stem cell is a mammalian primary cell.

90. The method of claim 89, wherein the mammalian primary cell is a human primary cell.

91. The method of claim 3, wherein the cell to which the RTA is administered is selected from the group consisting of a mesenchymal stem cell, a liver cell, a muscle cell, an osteoblast, a Schwann cell, an adipocyte, and a pre-adipocyte.

92. The method of claim 3, wherein the RTA is a protease inhibitor.

93. The method of claim 3, wherein the RTA is a NRTI.

94. The method of claim 3, wherein the culture conditions comprise culturing the cell in the presence of a receptor ligand selected from the group consisting of a PPARγ ligand, a RXR ligand, a retinoic acid receptor ligand, insulin, an insulin-like growth factor, a glucocorticoid receptor ligand, and a cAMP-elevating agent.

95. The method of claim 94, wherein the receptor ligand is a PPARγ ligand.

96. The method of claim 95, wherein the PPARγ ligand is an agonist of PPARγ.

97. The method of claim 96, wherein the PPARγ agonist is a thiazolidinedione.

98. The method of claim 94, wherein the receptor ligand is a RXR ligand.

99. The method of claim 98, wherein the RXR ligand is an agonist of RXR.

100. The method of claim 99, wherein the RXR agonist is LGD1069, LG100268, 9-cis retinoic acid, or all-trans retinoic acid.

101. The method of claim 94, wherein the receptor ligand is a retinoic acid receptor ligand.

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102. The method of claim 101, wherein the retinoic acid ligand is CH55, 9-cis retinoic acid, or all-trans retinoic acid.

103. The method of claim 94, wherein the receptor ligand is insulin.

104. The method of claim 94, wherein the receptor ligand is an insulin-like growth factor.

105. The method of claim 92, wherein the protease inhibitor is an aspartyl protease inhibitor.

106. The method of claim 105, wherein the aspartyl protease inhibitor is a viral aspartyl protease inhibitor.

107. The method of claim 106, wherein the viral aspartyl protease inhibitor is an HIV protease inhibitor.

108. The method of claim 93, wherein the NRTI is an HIV NRTI.

109. The method of claim 91, wherein the mesenchymal stem cell has the characteristics of a C3H10T1/2 cell.

110. The method of claim 109, wherein the mesenchymal stem cell is a mammalian primary cell.

111. The method of claim 110, wherein the mammalian primary cell is a human primary cell.

112. The method of claim 4, wherein the cell to which the RTA is administered is selected from the group consisting of a mesenchymal stem cell, a liver cell, a muscle cell, an osteoblast, a Schwann cell, an adipocyte, and a pre-adipocyte.

113. The method of claim 4, wherein the RTA is a protease inhibitor.

114. The method of claim 4, wherein the RTA is a NRTI.

115. The method of claim 4, wherein the culture conditions comprise culturing the cell in the presence of a receptor ligand selected from the group consisting of a PPARγ ligand, a RXR ligand, a retinoic acid receptor ligand, insulin, an insulin-like growth factor, a glucocorticoid receptor ligand, and a cAMP-elevating agent.

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116. The method of claim 115, wherein the receptor ligand is a PPARγ ligand.

117. The method of claim 116, wherein the PPARγ ligand is an agonist of PPARγ.

118. The method of claim 117, wherein the PPARγ agonist is a thiazolidinedione.

119. The method of claim 115, wherein the receptor ligand is a RXR ligand.

120. The method of claim 119, wherein the RXR ligand is an agonist of RXR.

121. The method of claim 120, wherein the RXR agonist is LGD1069, LG100268, 9-cis retinoic acid, or all-trans retinoic acid.

122. The method of claim 115, wherein the receptor ligand is a retinoic acid receptor ligand.

123. The method of claim 122, wherein the retinoic acid ligand is CH55, 9-cis retinoic acid, or all-trans retinoic acid.

124. The method of claim 115, wherein the receptor ligand is insulin.

125. The method of claim 115, wherein the receptor ligand is an insulin-like growth factor.

126. The method of claim 113, wherein the protease inhibitor is an aspartyl protease inhibitor.

127. The method of claim 126, wherein the aspartyl protease inhibitor is a viral aspartyl protease inhibitor.

128. The method of claim 127, wherein the viral aspartyl protease inhibitor is an HIV protease inhibitor.

129. The method of claim 114, wherein the NRTI is an HIV NRTI.

130. The method of claim 112, wherein the mesenchymal stem cell has the characteristics of a C3H10T1/2 cell.

131. The method of claim 130, wherein the mesenchymal stem cell is a mammalian primary cell.

132. The method of claim 131, wherein the mammalian primary cell is a human primary cell.

133. The method of claim 35, wherein the compound is screened for potential protease inhibitor activity.

134. The method of claim 35, wherein the receptor ligand is a PPARγ ligand.

135. The method of claim 134 wherein the PPARγ ligand is a thiazolidinedione.

136. The method of claim 134, wherein the ligand is BRL49653.

137. The method of claim 36, wherein the compound is screened for potential protease inhibitor activity.

138. The method of claim 36, wherein the receptor ligand is a PPARγ ligand.

139. The method of claim 138, wherein the PPARγ ligand is a thiazolidinedione.

140. The method of claim 138, wherein the ligand is BRL49653.

141. The method of claim 41, wherein the RTA is a protease inhibitor.

142. The method of claim 41, wherein the mammal is maintained under high-fat diet conditions.

143. The method of claim 41, wherein the mammal is a mouse.

144. The method of claim 143, wherein the mouse has the obesity-related characteristics of a AKR/J mouse.

145. The method of claim 43, wherein the RTA is a protease inhibitor.

146. The method of claim 43, wherein the mammal is maintained under high-fat diet conditions.

147. The method of claim 43, wherein the mammal is a mouse.

148. The method of claim 147, wherein the mouse has the obesity-related characteristics of a AKR/J mouse.

149. The method of claim 47, wherein the RTA is a protease inhibitor.

150. The method of claim 47, wherein the mammal is maintained under high-fat diet conditions.

151. The method of claim 47, wherein the mammal is a mouse.

152. The method of claim 151, wherein the mouse has the obesity-related characteristics of a AKR/J mouse.

153. The method of claim 48, wherein the RTA is a protease inhibitor.

154. The method of claim 48, wherein the mammal is maintained under high-fat diet conditions.

155. The method of claim 48, wherein the mammal is a mouse.

156. The method of claim 155, wherein the mouse has the obesity-related characteristics of a AKR/J mouse.

157. The method of claim 48, wherein the retinoid-activated gene is a gene which encodes alkaline phosphatase.

158. The method of claim 48, wherein the retinoid-activated gene is activated by a retinoid nuclear receptor.

159. The transgenic mouse of claim 55, wherein the RTA is a protease inhibitor.

160. The transgenic mouse of claim 56, wherein the RTA is a protease inhibitor.

161. The method of claim 58, wherein the RTA is an HIV protease inhibitor.

162. The method of claim 58, wherein the gene is a retinoid-activated gene.

163. The method of claim 58, wherein the gene is activated by a retinoid nuclear receptor.

164. The method of claim 58, wherein the gene is a PPARγ:RXR-activated gene.

165. The method of claim 58, wherein the gene is a protease inhibitor regulated gene.

166. The method of claim 58, wherein the change in gene expression comprises an increase in gene expression.

167. The method of claim 58, wherein the change in gene expression comprises a decrease in gene expression.

168. The method of claim 60, wherein the RTA is an HIV protease inhibitor.

169. The method of claim 60, wherein the gene is a retinoid-activated gene.

170. The method of claim 60, wherein the gene is activated by a retinoid nuclear receptor.

171. The method of claim 60, wherein the gene is a PPARγ:RXR-activated gene.

172. The method of claim 60, wherein the gene is a protease inhibitor regulated gene.

173. The method of claim 60, wherein the change in gene expression comprises an increase in gene expression.

174. The method of claim 60, wherein the change in gene expression comprises a decrease in gene expression.

175. The method of claim 62, wherein the RTA is an HIV protease inhibitor.

176. The method of claim 62, wherein the gene is a retinoid-activated gene.

177. The method of claim 62, wherein the gene is activated by a retinoid nuclear receptor.

178. The method of claim 62, wherein the gene is a PPARγ:RXR-activated gene.

179. The method of claim 62, wherein the gene is a protease inhibitor regulated gene.

180. The method of claim 62, wherein the change in gene expression comprises an increase in gene expression.

181. The method of claim 62, wherein the change in gene expression comprises a decrease in gene expression.